ARIC Manuscript Proposal # 920

PC Reviewed: 02/18/03	**Status: __A__**	**Priority: __2__**
SC Reviewed: 03/03/03	**Status: __A__**	**Priority: __2__**

1.a. Full Title: Psychosocial Factors as Predictors of ABI Change

b. Abbreviated Title (Length 26 characters):

2. Writing Group (list individual with lead responsibility first):

Keattiyoat Wattanakit (lead), Janice Williams, Pam Schreiner, Alan Hirsch, Aaron Folsom
Address: University of Minnesota
WBOB, Room 300
1300 South 2nd Street
Minneapolis, MN 55454
Phone: 612-626-8873
E-mail: wattanakit@epi.umn.edu or folsom@epi.umn.edu

3. Timeline: Analysis will begin in January 2003, and final manuscript expected to be completed in September 2003.

4. Rationale:

There has been mounting evidence suggesting that depression and anger proneness are novel risk factors for cardiovascular disease incidence and survival. A meta-analysis concludes that depression is an independent risk factor for the development of coronary heart disease (CHD) in initially healthy people, conferring the relative risk of 1.64 for all studies.[1] Likewise, anger proneness, measured by the Spielberger Trait Anger Scale, places normotensive middle-aged men and women in the ARIC study at higher risk for the development of CHD.[2] One hypothesis that might explain the association between depression or anger proneness and CHD involves the heightened sympathetic arousal and catecholamine secretion, resulting in increased platelet activation, hemodynamic changes, and disruption or progression of atherosclerotic plaque.[3,4]

Peripheral vascular disease (PVD) is an atherosclerotic disease in which the arterial blood supply does not meet the metabolic demand of the muscles on the lower extremity. It is known that patients with PVD not only suffer from physical disability, but also from psychosocial and emotional dysfunction.[5] Specifically they often experience a sense of shame and powerlessness, feeling of depression, inadequacy, and frustration, and poor general health and social function.[6,7] Despite of these consequences, there is little evidence showing psychosocial factors as the causes of PVD progression over time.

5. Main Hypothesis/Study Questions:

The main hypothesis to be tested is that depression, anger proneness, and poor social support are inversely associated with change in ABI. We hypothesize a dose-response relation for each

variable, and this relation will persist after adjusting for the traditional risk factors. Furthermore, it is speculated that participants who have high score when combining depression, anger proneness, and poor social support will have the steepest decline of ABI, and the worst symptom of leg claudication.

6. Data (variables, time window, source, inclusions/exclusions):

Independent variables: the Vital Exhaustion questionnaire, the Spielberger Trait Anger Scale, and the Luben Social Network Scale from visit 2
Dependent variables: change of ABI across visits 1, 3, and 4, and the Rose questionnaire
Covariates: diabetes, LDL, HDL, triglycerides, smoking, hypertension, Lp (a), fibrinogen, age, race, gender, and clinical site

Using the available standardized measurements from visit 2 as baseline, the association between the psychosocial factors and the progression of PVD will be assessed. The Vital Exhaustion questionnaire, the Spielberger Trait Anger Scale, and the Luben Social Network Scale will capture depressive symptom, anger proneness, and the quality of social support, respectively. Participants with PVD will be defined by low ABI measurement at visit 1 and response from the Rose questionnaire, and progression by corresponding changes in ABI through visits 3 and 4, Rose questionnaire, and revascularization. ABI change will be modeled by linear regression. Time-dependent ABI levels will be modeled by repeated measures regression with SAS's PROC Mixed. The adjusted relative risk for a large decline in ABI will be calculated for categories of depressive symptoms, anger proneness, and quality of social support using logistic regression, or if appropriate, Cox proportional hazard regression models using a dichotomous outcome, censoring once a threshold of ABI is crossed. To further explore the interaction among these psychosocial factors, a series of multivariate models will be constructed.

7.a. Will the data be used for non-CVD analysis in this manuscript? ____ Yes __x__ No

b. If Yes, is the author aware that the file ICTDER02 must be used to exclude persons with a value RES_OTH = "CVD Research" for non-DNA analysis, and for DNA analysis RES_DNA = "CVD Research" would be used? ____ Yes ____ No
(This file ICTDER02 has been distributed to ARIC PIs, and contains the responses to consent updates related to stored sample use for research.)

8.a. Will the DNA data be used in this manuscript? ____ Yes __x__ No

8.b. If yes, is the author aware that either DNA data distributed by the Coordinating Center must be used, or the file ICTDER02 must be used to exclude those with value RES_DNA = "No use/storage DNA"? ____ Yes ____ No

9. The lead author of this manuscript proposal has reviewed the list of existing ARIC Study manuscript proposals and has found no overlap between this proposal and previously approved manuscript proposals either published or still in active status.
ARIC Investigators have access to the publications lists under the Study Members Area of the web site at: http://bios.unc.edu/units/cscc/ARIC/stdy/studymem.html

___x___Yes _____ No

10. What are the most related manuscript proposals in ARIC (authors are encouraged to contact lead authors of these proposals for comments on the new proposal or collaboration)?

Williams J, Paton C, Siegler I, et al. Anger proneness predicts coronary heart disease risk: prospective analysis from the ARIC study. Circulation 2000;101:2034-2039.

References

1 Rugulies R. Depression as a predictor for coronary heart disease: a review and meta-analysis. Am J Prev Med 2002;23(1):51-61.

2 Williams J, Paton C, Siegler I, et al. Anger proneness predicts coronary heart disease risk: prospective analysis from the ARIC study. Circulation 2000;101:2034-2039.

3 Musselman DL, Tomer A, Manatunga AK, et al. Exaggerated platelet reactivity in major depression. Am J Psychiatry 1996;153:1313-1317.

4 Muller JE, Abela GS, Nesto RW, et al. Triggers, acute risk factors and vulnerable plaques: the lexicon of a new frontier. J Am Coll Cardiol 1994:23:809-813.

5 Treat-Jacobson D, Halverson S, Ratchford A, et al. A patient-derived prospective of health-related quality of life with peripheral arterial disease. J Nursing Schol 2002;34:155-160.

6 Hallin A, Bergqvist D, Fugl-Meyer K, et al. Areas of concern, quality of life and life satisfaction in patients with peripheral vascular disease. Eur J Vasc Endovasc Surg 2002;24:255-263.

7 Gibson J, Kenrick M. Pain and powerlessness: the experience of living with peripheral vascular disease. J Adv Nursing 1998;27(4):737-745.